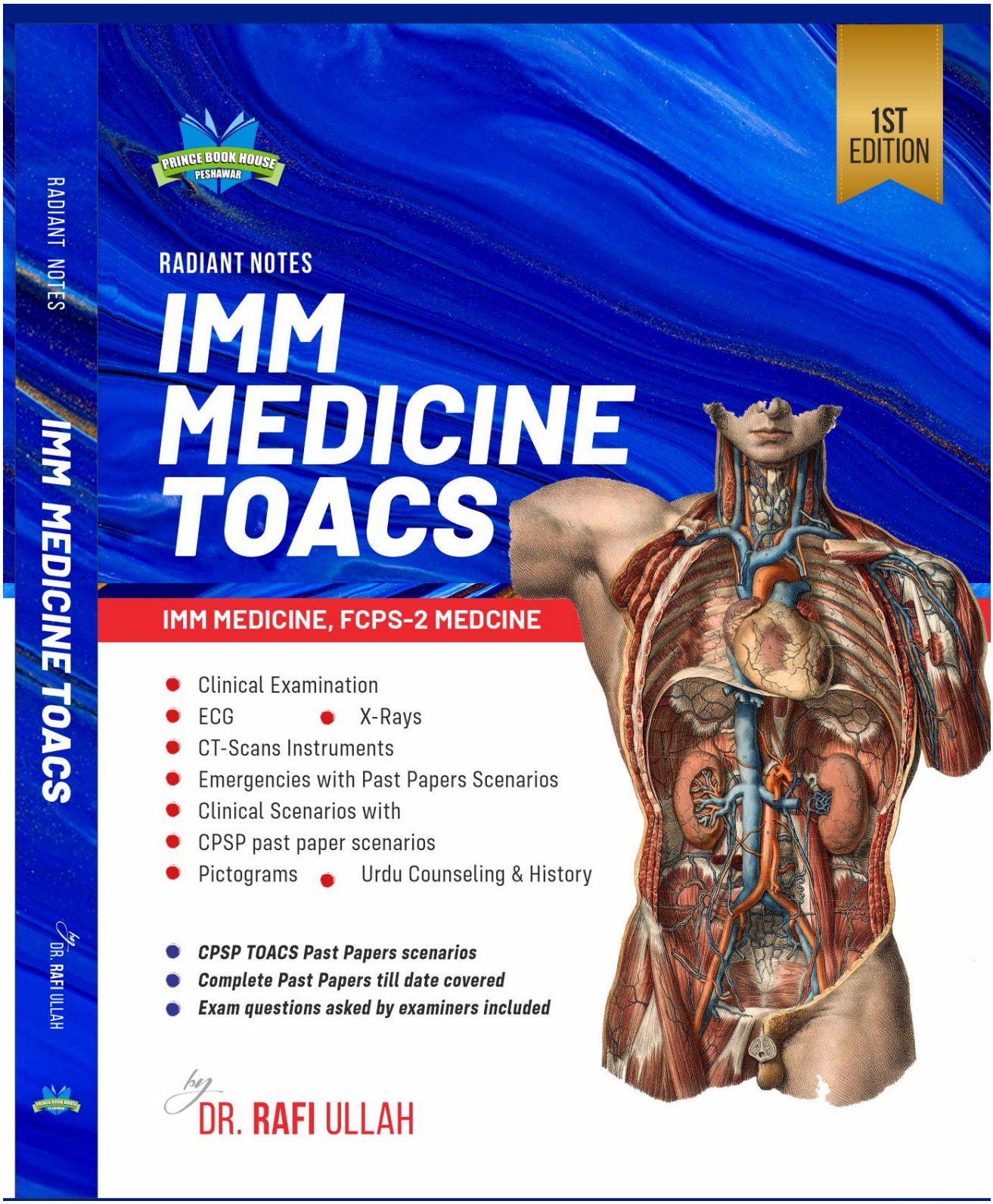
Sample pages from Our Book "IMM Medicine TOACS"



# Preface

- I am thankful to Almighty Allah who gave me the courage to write this book.

- Passing FCPS or others license examinations is a difficult milestone for many doctors, but is a mandatory requirement for career progression. Due to busy schedule of Doctors it is almost impossible to go through all the large text books so as to acquire all the knowledge that is required to pass the examinations.

- The aim of this book is to provide the busy doctor with a comprehensive review of all the text books

- This book is the only complete book for CPSP IMM TOACS which contains all the previous Past papers of CPSP IMM TOACS exam till date and real scenarios of exam

- CPSP TOACS scenarios and all stations explained separately with colored diagrams, pictograms, tables and easy format

- This book has been written to meet the needs and requirements of students appearing in IMM medicine and as well as FCPS-II Medicine exam.

- You won't have to read any other book---It includes examinations, ECG, clinical scenarios (with CPSP past paper scenarios), emergencies, poisoning, animal bites, Instruments, pictograms, Urdu counselling & Urdu histories and much more

  - Any suggestions/ corrections would be highly appreciated or you can send us your exam questions and his/her name will appear in the future edition. For which you can contact us on our page or email ID

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# Station Examinations

- ***Previous TOACS station and their answers given with each system***

# Examination: 1 General Physical

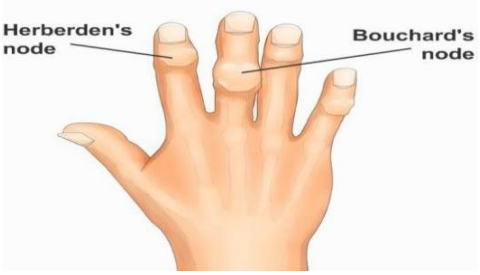
Common Command:

- Perform the general physical examination of this patient and describe only positive findings.
- And then question depends upon your findings

Important TIP:

- Watch YouTube videos on examination, practice as much as you can along with the timer, try to perform examination in 2 minutes---so that you can answer the examiner questions then.

## General Physical Examination

| | |
|---|---|
| **The first & most important step** | ❖ **If there is a sanitizer beside patient, make sure to use that**<br>❖ **Be sure that you are on the right side of the patient**<br>❖ At the same time say **greetings, introduce yourself, take permission and explain** what you are going to do<br>❖ The patient legs should be exposed till knees |
| **Observation and Appearance** | ❖ Before starting examination take a few moments to quickly look from head to toe—while standing at the foot side and look for<br>○ General appearance (whether he looks well, mildly ill or severely ill)<br>○ Consciousness (e.g. alert, confused, drowsy, unconscious)<br>○ Look for surrounding findings (e.g., IV canula is attached on right/Left hand, any chest tube or foleys catheter)<br><br>**Example: An old ill looking man lying in the bed, of average height and built, he is fully conscious with an IV canula in left hand** |
| **Hand** | ❖ **General look at the hand---remember to observe both hands at the same time and compare both of them as well**<br>○ Shape of the hand---short 4th or 5th metacarpals in pseudohypoparathyroidism<br>○ Carpal spasm in tetany<br>○ Hands are larger in acromegaly<br>○ Tremors ---ask the patient to outstretch the arms and abduct the fingers<br>❖ **Nails**<br>○ Look for Pale nails (Anemia, liver disease), white nails with darker rims (Hepatitis), Bluish nails (Cyanosis), Splinter haemorrhages (Infective endocarditis), Pitting of nails (Psoriasis), Koilonychias (spoon-shaped nails----seen in long standing iron deficiency anemia)<br>○ Clubbing When two fingers are approximated normally there is a space btw the two nails. In clubbing this is absent (Schamroth's sign). Clubbing simply means loss of angle, Seen in diseases of heart and lungs<br>○ Clubbing is explained later<br><br>❖ **Examination of fingers**<br>○ Osler's nodes---pea size painful swelling in the pulps of terminal phalanges seen in infective endocarditis<br>○ Heberden's nodes --------bony swellings<br>○ Bouchards nodes<br><br>  |

| | |
|---|---|
| | ❖ **Examination of The Hands ad palms**<br>○ Pallor: in anemia<br>○ Cyanosis: CVS and respiratory disease<br>○ Sweating – excessive sweating may be idiopathic, but is also seen in anxiety (palm is cold and sweaty), and thyrotoxicosis (palm is warm and sweaty)<br>○ Dupuytren's contracture ---there is thickening of palmar fascia felt as thickened plaque or cord between palm and ring and little finger ---seen in alcoholic cirrhosis<br>○ Palmar erythema: reddening of the palms at thenar and hypothenar eminence. Seen in chronic hepatitis and portal HTN. |
| **Vital Signs Examination** | ❖ It includes:<br>○ Temperature<br>○ Respiratory rate<br>○ Pulse rate<br>○ Blood pressure<br><br>**Few Points to Be Remembered**<br>- Start by placing a thermometer in axilla for a minute<br>- During this minute examine the pulse for 30 secs if regular or for a full one minute if irregular ---at the end compare pulse in left hand as well as femoral---how and what to look for pulse is explained later<br>- During this one minute----also count respiratory rate<br>- After this---remove the thermometer, and then check the Blood pressure (How to check BP is given separately in CVS) |
| **Examination of Face** | ❖ General appearance<br>○ Characteristics facies e.g. moon like face in Cushing, mask like or expressionless face in parkinsonism<br>❖ Look for puffiness<br>○ Seen in renal failure, nephrotic syndrome, nephritic syndrome, angioedema<br>❖ Eyes:<br>○ Look for any infection, redness and a quick general look<br>○ Exophthalmos---it means protrusion of eyeball seen in Grave's disease<br>○ Xanthelasmas---yellow plaques on eyelids seen in hyperlipidemia<br>○ Conjunctiva --- check for anemia<br>✓ Ask the patient to look upwards, pull the lower eyelid downwards<br>✓ Remember check both eyes at the same time, use both hands<br>○ Yellow discoloration of sclera for jaundice<br>✓ Ask the patient to look downwards and pull the upper eyelid upwards<br>✓ Normal its white, but in jaundice becomes yellow<br>✓ Remember: use your left hand to pull the upper eyelids and use your right hand as an object to which patient should look at. Check both eyes at the same time<br>❖ **Cheeks:**<br>○ In SLE there is a rash over check and bridge of nose (butterfly rash)<br>❖ **Nose:**<br>○ Look for any discharge, symmetry, deviated nasal septum<br>❖ Bluish discoloration of the tip of nose and ear lobules for cyanosis<br>❖ Tongue:<br>○ Dryness, pallor and cyanosis of dorsum of tongue<br>○ Yellowness of the undersurface of the tongue |
| **Examination of The Neck** | ❖ Examination of thyroid (discussed separately)<br>❖ Examination of JVP (discussed separately)<br>❖ Examination of lymph nodes<br>○ Neck lymph nodes<br>✓ Submental (under the chin)<br>✓ Submandibular (under the jaw)<br>✓ Pre and post auricular<br>✓ Occipital |

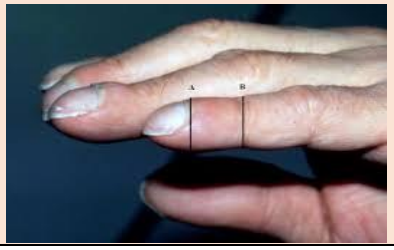
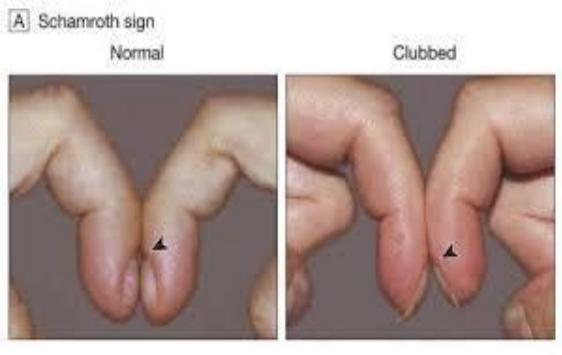
| | |
|---|---|
| | ✓ Lymph nodes of posterior triangle behind the Sternocleidomastoid<br>✓ Lymph nodes of Anterior triangle in front of Sternocleidomastoid<br>○ Axillary lymph nodes<br>✓ <u>Right axilla</u><br>• Lymph nodes of apical central and medial group----------Elevate the patient arm above his head **with right hand and push fingers of left hand** up in axilla, palm facing patient's chest, bring back patients arm alongside his chest<br>• Lymph nodes of anterior group -------For palpation of anterior group hold anterior axillary folds between thumb and fingers of your left hand<br>• Lymph nodes of Lateral group -------place palmar aspect of **fingers of your right hand** along the medial side of humerus<br>• Lymph nodes of Posterior group---hold posterior axillary folds between thumb and fingers of your corresponding hand from behind the patient<br>✓ <u>Left axilla</u><br>• Same process is repeated<br>• Apical central and medial groups are **palpated with right hand**<br>• While lateral group **palpated with left hand**<br>○ <u>Inguinal lymph nodes</u><br>• Palpable over the inguinal ligament |
| **Examination of Feets** | ❖ Look for clubbing, cyanosis and koilonychia in feet as well<br>❖ Feet are commonly affected by ischemia due to peripheral vascular disease, early signs are loss of hair and shimmy skin<br>❖ Look for edema<br>○ Look for edema on dorsum of the foot and shin<br>○ In a bed ridden patient also check over the sacrum<br>○ **Compare both legs while examining**<br>○ Press the thumb for at least 5 seconds |
| **Last and most important step** | ❖ **Say thank you to the patient and cover the patient** |

## Few Common Questions in General Physical Examination

### Que: 1 What are the causes of pitting and non-pitting edema?

| | Causes of Edema |
|---|---|
| **Pitting edema** | **Generalized / bilateral**<br>❖ Cardiovascular system<br>○ Right heart failure, constrictive pericarditis, IVC obstruction<br>❖ Renal failure<br>❖ Nephrotic syndrome<br>❖ Liver cirrhosis<br>❖ Malnutrition<br>❖ Malabsorption<br><br>**Localized**<br>❖ Immobilization<br>❖ Venous obstruction |
| **Non-pitting edema** | ❖ Lymphatic obstruction<br>❖ Angioedema |

## Que: 2 How to check clubbing, its causes and degree of clubbing?

| | |
|---|---|
| **Definition** | ❖ Bulbous enlargement of the ends of one or more fingers or toes<br>  |
| **Grades of Clubbing** | ❖ Stage 1:<br>○ Fluctuation of nail bed<br>❖ Stage 2:<br>○ Loss of the normal <165° angle (Lovibond angle) between the nail-bed and the fold (Schamroth's window is obliterated. Clubbing is not obvious at a glance).<br>❖ Stage 3:<br>○ Increased convexity of the nail fold. Clubbing is apparent at a glance.<br>❖ Stage 4:<br>○ Thickening of the whole distal (end part of the) finger (resembling a **drumstick**)<br>❖ Stage 5<br>○ Hypertrophic osteoarthropathy→ Shiny aspect and striation of the nail and skin |
| **Schamroth's Test or Schamroth's Window Test** | ❖ Place fingernails of same finger on opposite hands against each other, nail to nail.<br>❖ A small diamond-shaped "window" is normally apparent between the nail-beds.<br>❖ If this window is obliterated, the test is positive and clubbing is present.<br>  |
| **Causes of Clubbing** | Pulmonary:<br>❖ Lung CA, bronchiectasis, pulmonary fibrosis, abscess, CF, empyema<br>Cardiac<br>❖ Cyanotic heart disease, endocarditis, A-V fistula<br>Gastrointestinal<br>❖ IBD, celiac, cirrhosis<br>Endocrine<br>❖ Graves<br>Others<br>❖ Malignancy, Primary hypertrophic osteoarthropathy |
| **Clinical Pearls** | **Clubbing is not seen in COPD – if present, think malignancy**<br>**Differential clubbing -----it means clubbing in the toes but not in fingers e.g. in PDA with revered shunt** |

## Que: 3 What are the causes of palpable lymph nodes?

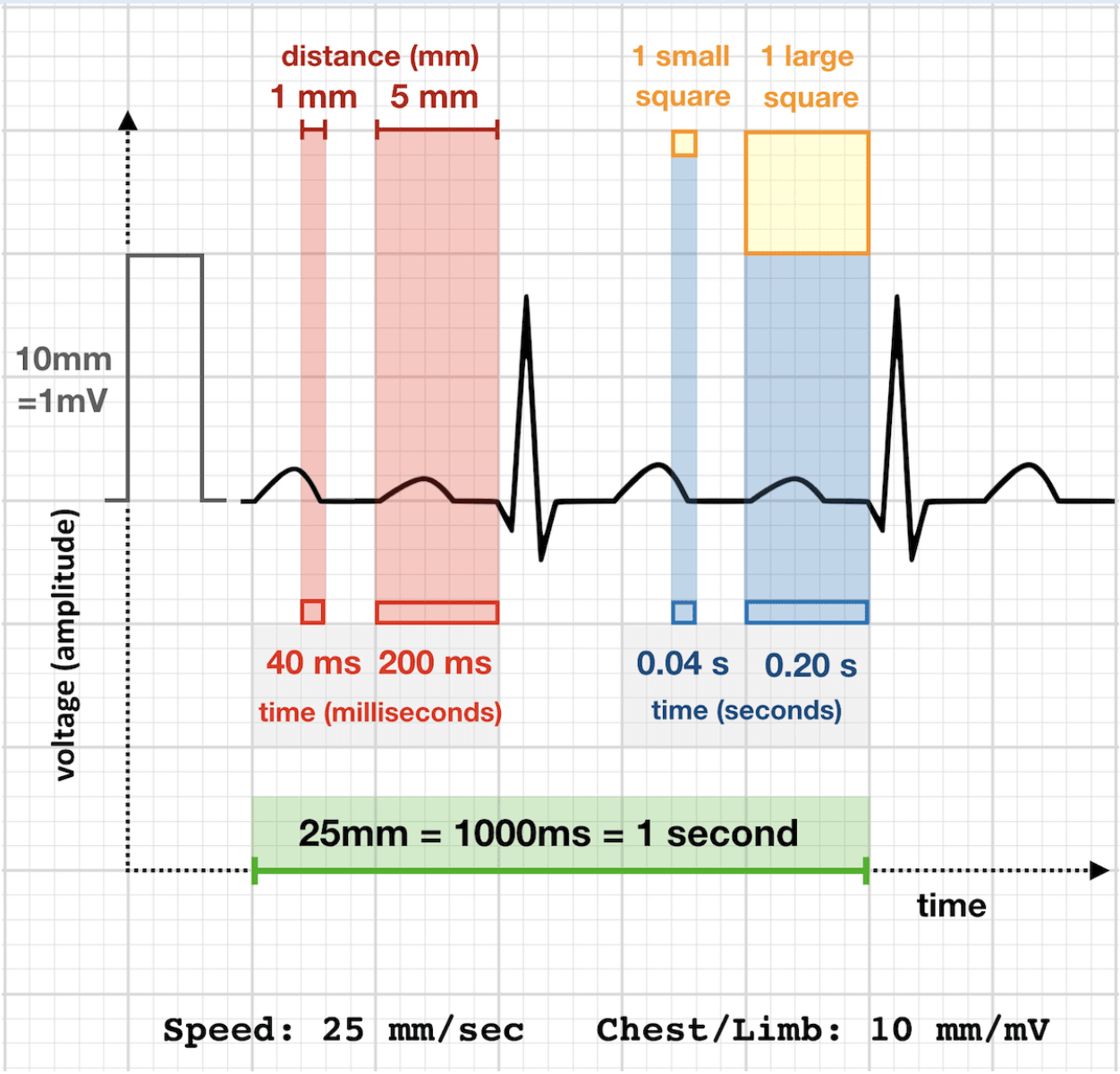
| | |
|---|---|
| **Infections** | ❖ Viral<br>  ○ HIV, EBV, CMV.<br>❖ Bacterial<br>  ○ Brucellosis, leptospirosis, TB, Streptococcal.<br>❖ Protozoal<br>  ○ Toxoplasmosis<br>❖ Fungal<br>  ○ Histoplasmosis & Coccidioidomycosis |
| **Immunologic** | ❖ Collagen vascular disease, Drug hypersensitivity (e.g., phenytoin), serum sickness |
| **Neoplasm** | ❖ Lymphoma, leukemia, amyloidosis, metastatic carcinoma |
| **Other** | ❖ Sarcoidosis; lipid storage diseases |
| **Factors That Favor Biopsy** | ❖ Age (40 Y)<br>❖ Size (2 Cm)<br>❖ Location (supraclavicular is always abnormal)<br>❖ Duration (1 Month). |
| **Characteristic Findings of lymph nodes** | ❖ Matted lymph nodes<br>  ✓ TB (most common)<br>  ✓ Lymphoma<br>  ✓ Actinomycosis<br>❖ Hard lymph nodes<br>  ✓ Malignancy<br>❖ Tender lymph nodes<br>  ✓ Acute inflammation (may be secondary to mastoiditis, tonsilitis)<br>  ✓ Infection of lymph nodes itself<br>❖ Lymphadenopathy with sinus<br>  ✓ Tuberculosis |

## Pigmentation

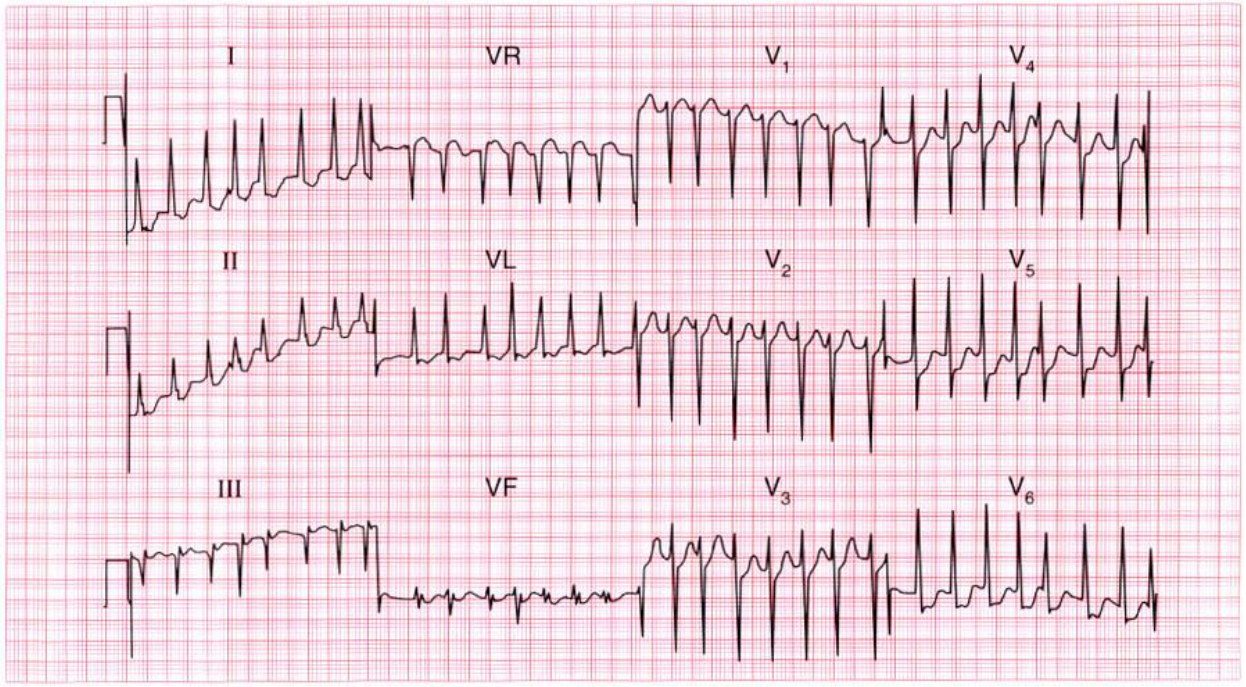
| | |
|---|---|
| **Causes of pigmentation** | ❖ Physiological<br>  ○ Familial, pregnancy & sunbath<br>❖ Pathological<br>  ○ Endocrine causes:<br>  ▪ Addison, Cushing, Nelson syndrome<br>  ○ Infections<br>  ▪ Kala Azar<br>  ○ CLD:<br>  ▪ Hemochromatosis, primary biliary cirrhosis<br>  ○ GIT<br>  ▪ Whipple disease<br>  ○ Drugs<br>  ▪ Busulphan, amiodarone, Phenytoin and phenothiazines<br>  ○ Others:<br>  ▪ Chronic arsenic poisoning, |
| **Hints** | ❖ History of fever in kala azar<br>❖ Hepatosplenomegaly in kala azar<br>❖ BP (low in Addison)<br>❖ Abdomen (bilateral adrenalectomy scar in nelson syndrome)<br>❖ Evidence of other chronic illness |

# Station

# ECG


distance (mm)
1 mm
5 mm
1 small square
1 large square
10mm =1mV
voltage (amplitude)
40 ms
200 ms
time (milliseconds)
0.04 s
0.20 s
time (seconds)
25mm = 1000ms = 1 second
time
Speed: 25 mm/sec
Chest/Limb: 10 mm/mV

# Atrial Fibrillation




| | |
|---|---|
| **Interpretation** | The ECG shows:<br>❖ Hear rate—upto 200/min<br>❖ Absent P wave, Irregular R-R interval<br>❖ Normal axis<br>❖ Normal QRS complexes |
| **Diagnosis** | ❖ Atrial Fibrillation with rapid ventricular rate |
| **Causes** | (Mnemonic ---**C**(sea)-**PIRATES)**<br>❖ **C**HF<br>❖ **P**ulmonary embolism, **P**ost-operative (thoracic or cardiac surgery,)<br>❖ **I**schemic heart disease (Including MI), **I**diopathic (Lone atrial fibrillation)<br>❖ **R**heumatic heart disease (Mitral stenosis, Mitral regurgitation)<br>❖ **A**trial myxoma, **A**lcohol (excess and sudden withdrawal)<br>❖ **T**hyrotoxicosis (this condition should be excluded with the initial episode).<br>❖ **E**levated blood pressure (Hypertension), **E**lectrolytes disturbance (Dec K, Dec Mg)<br>❖ **S**leep apnea, **S**epsis |
| **Classification** | ❖ Paroxysmal AF→ that terminates spontaneously in <7 days and <48 hours in duration<br>❖ Persistent→ Sustained for >7 days, but can be terminated by chemical or electrical cardioversion<br>❖ Permanent→ Typically >1 y and when cardioversion has failed or in which clinical judgement has led to a decision not to pursue cardioversion |
| **Treatment** | ❖ Hemodynamically unstable patients<br>○ Hemodynamically unstable patients (e.g. shock or severe hypotension, pulmonary edema, or ongoing myocardial infarction) needs Urgent electrical cardioversion<br>○ Shock is administered in **synchrony with the R wave**.<br><br>❖ Hemodynamically stable patients<br>○ **Step: 1 Rate control:** Beta-blocker or calcium channel blocker (orally or intravenously) is usually the first-line agent, **Digoxin should be used if there is co-existing heart failure**<br>○ **Step 2: Prevention of thromboembolic complications** |

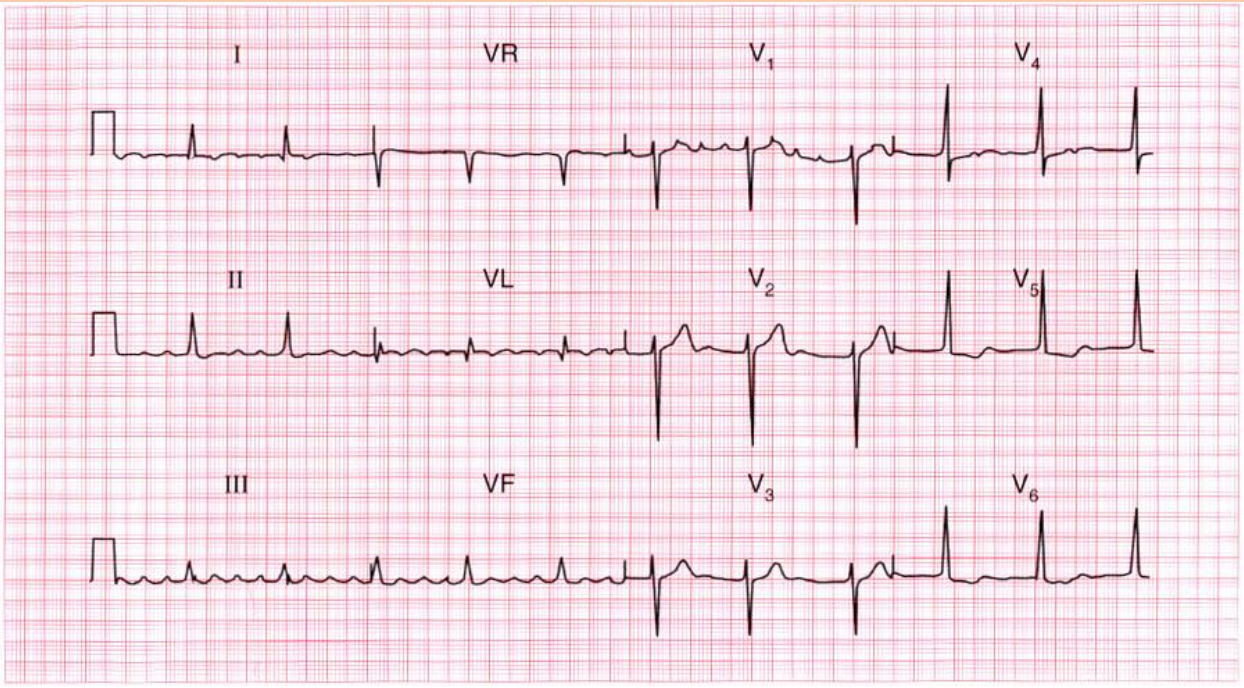
- Asses for the risk of thromboembolic events and decide for Anticoagulation by using $CHADS_2Vasc$ score

| **$CHA_2DS_2$-VASc score**<br>New recommended scoring system | | | |
|---|---|---|---|
| **C** | **C**HF or LVEF <40% | 1 | **Score = O** –no antithrombotic therapy |
| **H** | **H**ypertension | 1 | |
| **$A_2$** | **A**ge ≥75 years | **2** | |
| **D** | **D**iabetes mellitus | 1 | **Score= 1** ---Oral anticoagulation > anti-platelet therapy (If 1 score due to sex only then no antithrombotic |
| **$S_2$** | **S**troke or TIA | **2** | |
| **V** | **V**ascular disease (previous MI peripheral artery disease, or aortic plaque) | 1 | **Score ≥ 2=** Oral anticoagulation |
| **A** | **A**ge 65-74 years | 1 | |
| **S** | Female **S**ex | 1 | |

- **Step 3: Rhythm control & Anticoagulation**
  - **AF < 48 hours**
    - Intravenous heparin followed by cardioversion (electrical > pharmacological)
    - Electrical cardioversion or DC cardioversion---200-360J in synchrony with R wave
    - Pharmacological cardioversion----- can be done by Class III antiaryhtmatics (Mnemonic—**AIDS** ---- **A**miodarone, **I**butilide, **D**ofetilide, **S**otalol) and Class IC (flecainide, and propafenone)

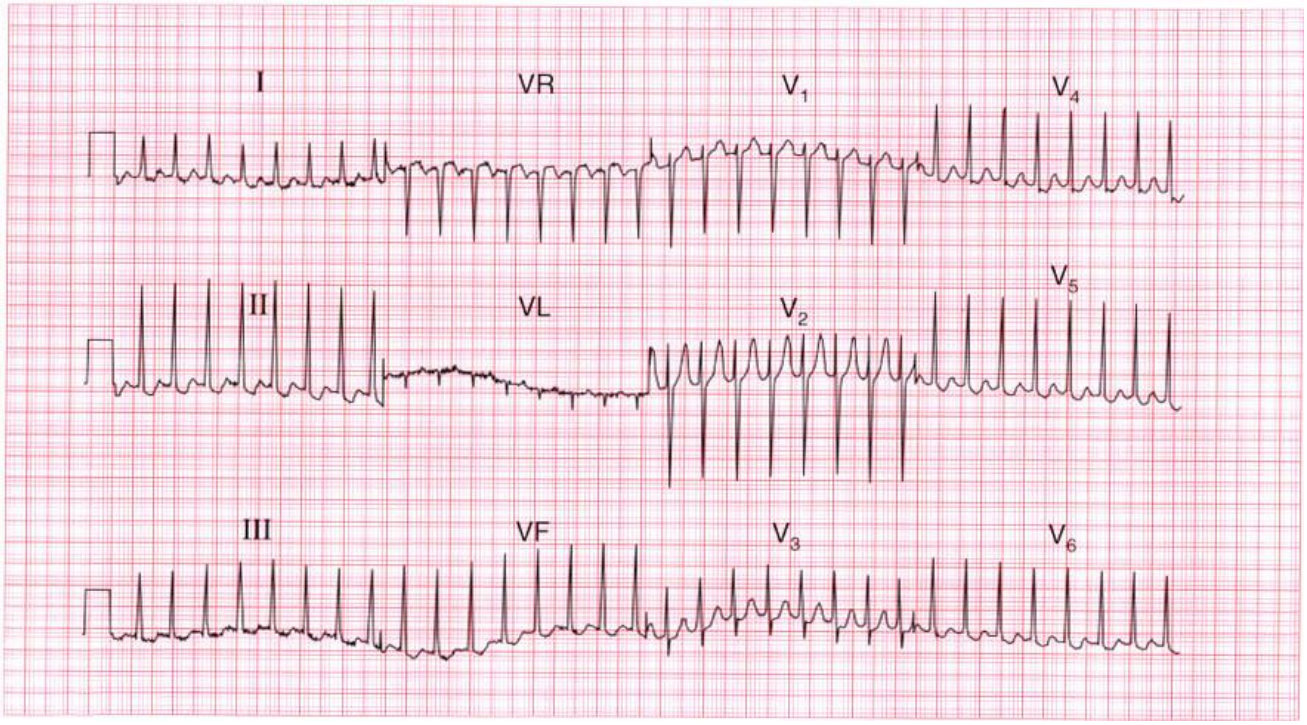
  - **AF > 48 hours (or time unknown) --------**Perform any of the following step
    - Step I-----Anticoagulate for 3 weeks with warfarin before performing cardioversion and for 4 weeks after performing cardioversion, target INR 2-3,
    - Step II----To avoid waiting for 3 weeks for anticoagulation, obtain trans-esophageal echocardiogram to look for left atrial thrombus
    - If thrombus is present then follow the step 1
    - If no thrombus—directly perform cardioversion without anticoagulation

# Atrial Flutter



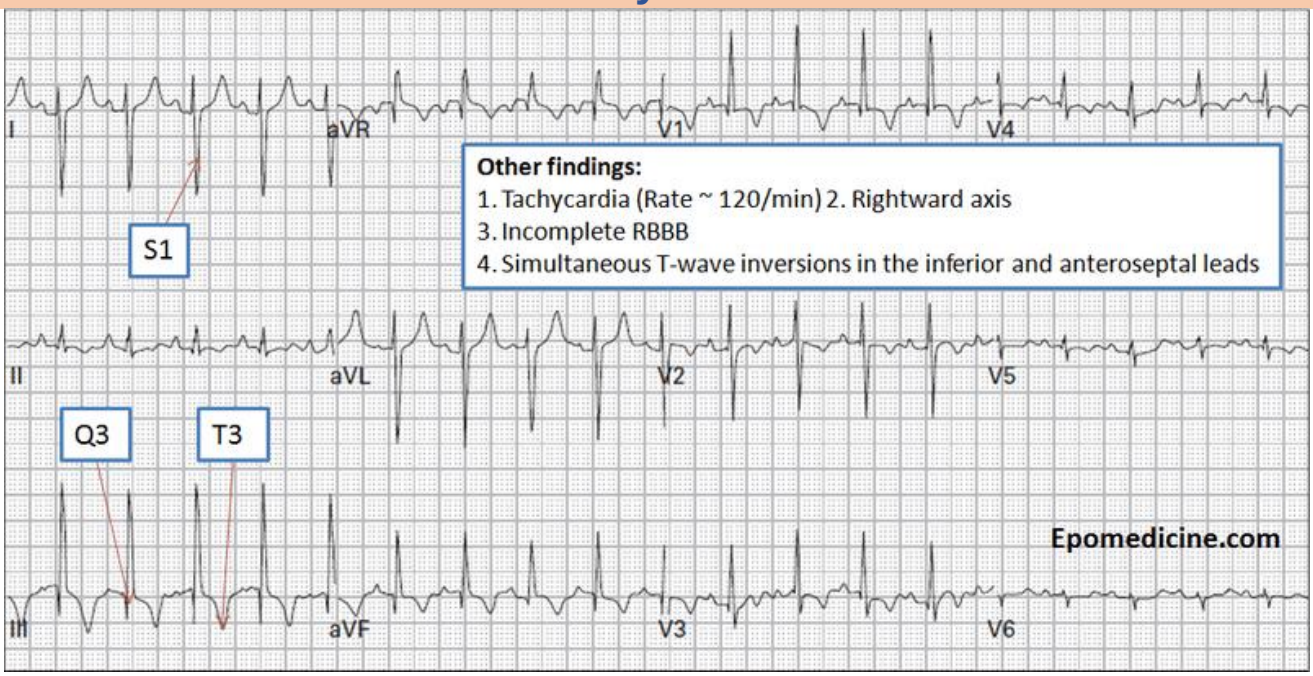

| | |
|---|---|
| **Interpretation** | The ECG shows:<br>❖ Ventricular rate—75/min, while atrial rate 280-300/min<br>❖ There are four P waves (one embedded in T wave at some areas) per QRS complex (arrowed)<br>❖ Normal axis<br>❖ Normal QRS complexes |
| **Diagnosis** | ❖ Atrial flutter 4:1 Block |
| **Causes** | ❖ CAD, thyrotoxicosis, mitral valve disease, cardiac surgery, COPD, PE, pericarditis |
| **Treatment** | ❖ Rate control = same agents as atrial fibrillation<br>❖ Rhythm control and anticoagulation<br>○ Class I antiaryhtmatics= results in slowing of AV conduction with subsequent hemodynamic collapse.<br>○ Class III antiaryhtmatics (Ibutilide and amiodarone) = preferred agents<br>○ Electrical cardioversion or DC cardioversion<br>• Synchronized shocks<br>• Risk of thromboembolism equal to atrial fibrillation<br>• Pre-cardioversion anticoagulation is not necessary for atrial flutter of less than 48 hours duration except in the setting of mitral valve disease<br>• Anticoagulation should be continued for at least 4 weeks after electrical or chemical cardioversion<br>❖ **Catheter ablation = treatment of choice** |

# Supraventricular Tachycardia (SVT)




| | |
|---|---|
| **Interpretation** | The ECG shows:<br>❖ Narrow complex tachycardia at 200/min<br>❖ No P waves visible<br>❖ Normal axis<br>❖ QRS complexes normal |
| **Diagnosis** | ❖ Supraventricular tachycardia |
| **Causes** | ❖ Physiological: Anxiety, tea, Alcohol.<br>❖ Thyrotoxicosis<br>❖ Ischemic heart disease<br>❖ Digitalis toxicity |
| **Treatment** | ❖ **Hemodynamically unstable---DC Cardioversion**<br>❖ **Hemodynamically Stable—**<br>○ Rest and assurance<br>○ Carotid sinus massage or Valsalva maneuver –it act by increasing the vagal tone<br>○ **If no response---**<br>▪ Adenosine ----6mg IV bolus into a large vein, followed by 0.9% saline flush, while recording a rhythm strip. If unsuccessful, after 2 min give 12mg, then one further 12mg bolus.<br>▪ Warn about SE: transient chest tightness, dyspnoea, headache, flushing.<br>▪ Relative CI: Asthma, 2nd/3rd-degree AV block or sinoatrial disease (unless pacemaker).<br>▪ If adenosine fails, use verapamil ~5mg IV over 2–3min. If no response, a further 5mg IV over 3min (if age <60yrs). Alternatives: atenolol 2.5mg IV repeated at 5min intervals until 10mg given; or amiodarone.<br>▪ If unsuccessful, use DC cardioversion. |

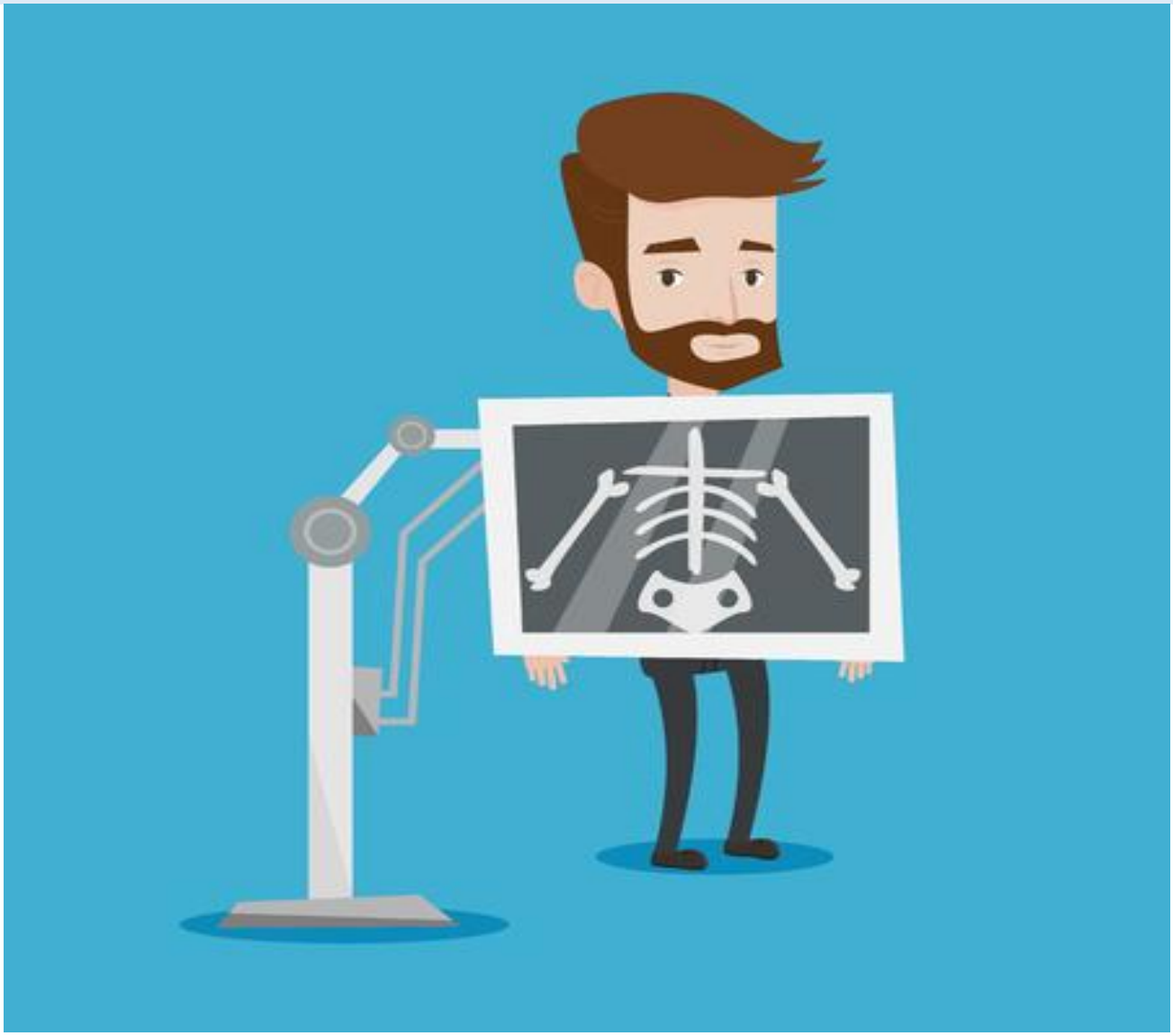
# Pulmonary Embolism



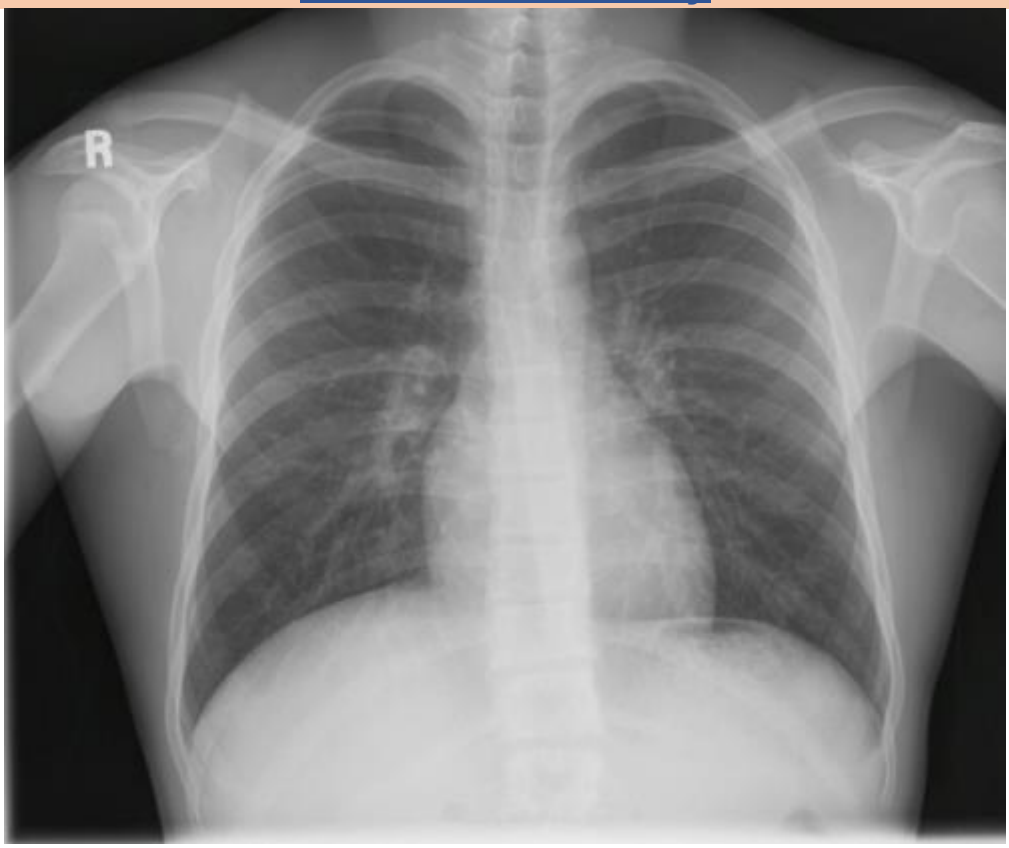

| | |
|---|---|
| **Interpretation** | The ECG shows:<br>❖ Sinus tachycardia<br>❖ Incomplete RBBB pattern<br>❖ S1 Q3 T3 pattern<br>❖ T wave inversion in lead v1-v4 |
| **Diagnosis** | ❖ Acute Pulmonary embolism |
| **Investigations** | ❖ Use Wells' criteria for PE to guide investigations<br>❖ D-dimer (considered positive >500ng/mL)- If positive do either CT-PA or V/Q scan<br>❖ CT-PA<br>o **First line and gold standard investigation**<br>o Requires administration of intravenous radiocontrast dye but is otherwise non-invasive<br>o Avoid in patients with renal impairment, and dye allerg<br>❖ V/Q scan:<br>o V/Q scanning requires administration of radioactive material Xenon gas (via both inhaled and IV routes<br>o Useful in patients with renal impairment and dye allergy<br>o It is most useful in patients without significant cardiopulmonary disease and a normal chest X-ray<br>❖ U/S (Doppler)<br>o Colour Doppler ultrasound of the leg veins remains the investigation of choice in patients with suspected DVT, but may also be applied to patients in whom PE is suspected (Approximately 50-70% of patients who have symptomatic pulmonary emboli will have lower extremity DVT when evaluated) |
| **Treatment** | ❖ Overlapping therapy of LMWH and warfarin<br>❖ Thrombolysis:<br>o First-line treatment for massive PE where there is circulatory failure (e.g. hypotension, acidosis).<br>o Streptokinase -----250,000 U as a loading dose over 30 minutes, followed by 100,000 U/hr over 12-24 hours. |
| **Duration of therapy:** | ❖ First episode of VTE due to reversible risk factors (e.g. surgery, major trauma) = 3 months<br>❖ If VTE is unprovoked i.e. idiopathic or risk factors are weak = 6 months<br>❖ Persistent prothrombotic risk or a history of previous emboli (second episode) = lifelong |

# Station

# X-Rays

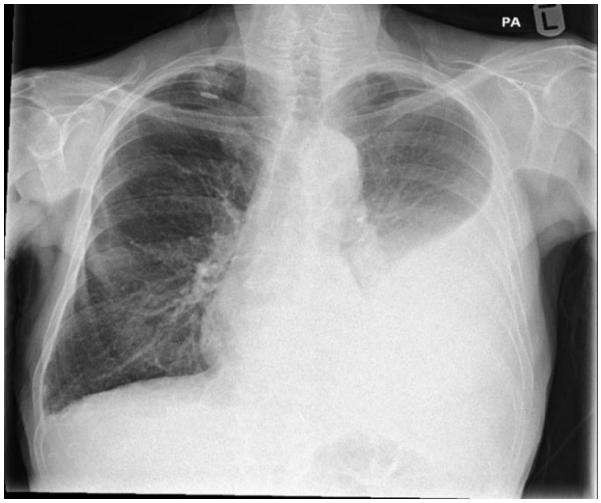


# Normal Chest X-Ray



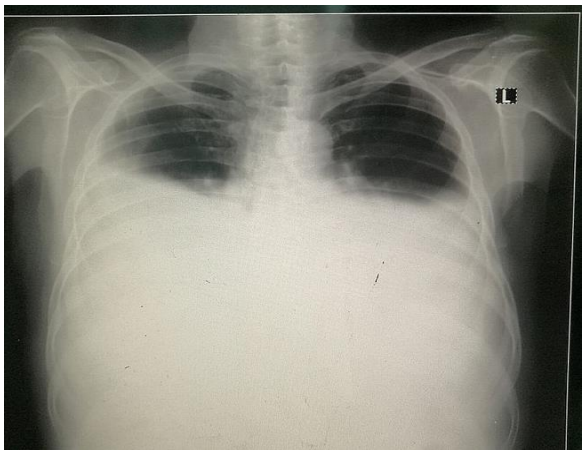

- **How to Report/ Represent Chest X-Ray**
  - Name of the X-ray (e.g. chest abdomen etc.)
  - View (e.g. AP, PA, Lateral view)
  - Check for rotation (ensure each clavicle is at the same distance to vertebrae)
  - **A**----**A**irway
    - Ensure trachea is visible and in midline
    - Check for tubes, pacemaker, lines, forign body etc.
    - Check for widened mediastinum
  - **B**-----**B**ones
    - Check for fracture
    - Also check soft tissue for subcutaneous air, foreign bodies and surgical clips
  - **C**----**C**ardiac
    - Check heart size and border
    - Check heart valves for calcification and valve replacements
  - **D**----**D**iaphragm
  - **E**----**E**ffusion
  - **F**----**F**ields—lung

# Pleural Effusion




| | |
|---|---|
| **Interpretation** | ❖ X-ray chest of patient PA view, taken on date XYZ, no bony pathology seen, no subcutaneous air, no visible line, tube or any bony pathology seen<br>❖ Homogenous opacity with concave upper margin on left side with shift of trachea to right side<br>❖ Obliteration of Costophrenic and Cardiophrenic angles on Left side<br>❖ Silhouette sign is positive |
| **Diagnosis** | ❖ Left sided pleural effusion |
| **Differential diagnosis** | ❖ All the causes of exudative effusion as given below |

# Bilateral Pleural Effusion




| | |
|---|---|
| **Interpretation** | ❖ X-ray chest of patient AP view, taken on date XYZ, no bony pathology seen, no subcutaneous air, no visible line, tube or any bony pathology seen<br>❖ Bilateral Homogenous opacity with concave upper margin<br>❖ Obliteration of Costophrenic and Cardiophrenic angles on both sides |
| **Diagnosis** | ❖ Bilateral pleural effusion |

# Toacs Related Question on Pleural Effusion

| Transudative Effusion | Exudative Effusion |
|---|---|
| ❖ Alteration of systemic factors that affect the formation and absorption of pleural fluid (e.g. Increased capillary hydrostatic pressure, decreased plasma oncotic pressure) | ❖ Increased permeability of pleural capillaries or lymphatic dysfunction |
| ❖ Usually bilateral | ❖ Can be bilateral or unilateral |
| ❖ Causes<br>○ Mnemonic: **S**ome **P**ersistent **P**eople **C**an **C**ause **N**ausea<br>▪ **S**VC syndrome<br>▪ **P**eritoneal dialysis<br>▪ **P**ulmonary embolism<br>▪ **C**HF (>90% cases—most common)<br>▪ **C**irrhosis<br>▪ **N**ephrotic syndrome | ❖ Causes:<br>○ Mnemonic: **PICTURE MAP**<br>▪ **P**arapneumonic effusion (associated with bacterial pneumonia, lung abscess)<br>▪ **I**nfections (viral, bacterial, fungal)<br>▪ **C**ancer<br>▪ **T**rauma/tumour (chylothorax)<br>▪ **U**remia<br>▪ **R**ickettsial infection<br>▪ **E**sophageal perforation<br>▪ **M**eigs syndrome (ascites +hydrothorax associated with ovarian tumor)<br>▪ **A**sbestos<br>▪ **P**ancreatic disease (elevated pleural fluid amylase) |

- **Distinguish clinically using Light's Criteria**

| Lights Criteria | | |
|---|---|---|
| | **Exudate** | **Transudate** |
| Pleural fluid protein **:** serum protein | >0.5 | <0.5 |
| Pleural fluid LDH: to serum LDH | >0.6 | <0.6 |
| Pleural LDH | >2/3 upper limit of N serum LDH | <2/3 upper limit of N serum LDH |
| Note:<br>All criteria for transudate must be fulfilled to be considered a transudative effusion.<br>If any one of the criteria for exudates is met – it is an exudate | | |

❖ **Clinical Features:**
- Often asymptomatic
- Dyspnea: varies with size of effusion and underlying lung function
- Pleuritic chest pain
- Inspection: trachea deviates away from effusion, ipsilateral decreased expansion
- Percussion: decreased tactile fremitus, dullness
- Auscultation: decreased breath sounds, bronchial breathing and egophony at upper level, pleural friction rub

❖ **Management:**
- Treat the underlying cause
- If the effusion is symptomatic do therapeutic thoracentesis and use the following step wise approach
  - Effusion resolved-----Observation
  - Effusion re-accumulates---Pleurodesis with talc or bleomycin or tetracycline
    - Pleurodesis successful--- observation
    - Pleurodesis unsuccessful----Consider pleurectomy, pleural abrasion or shunt

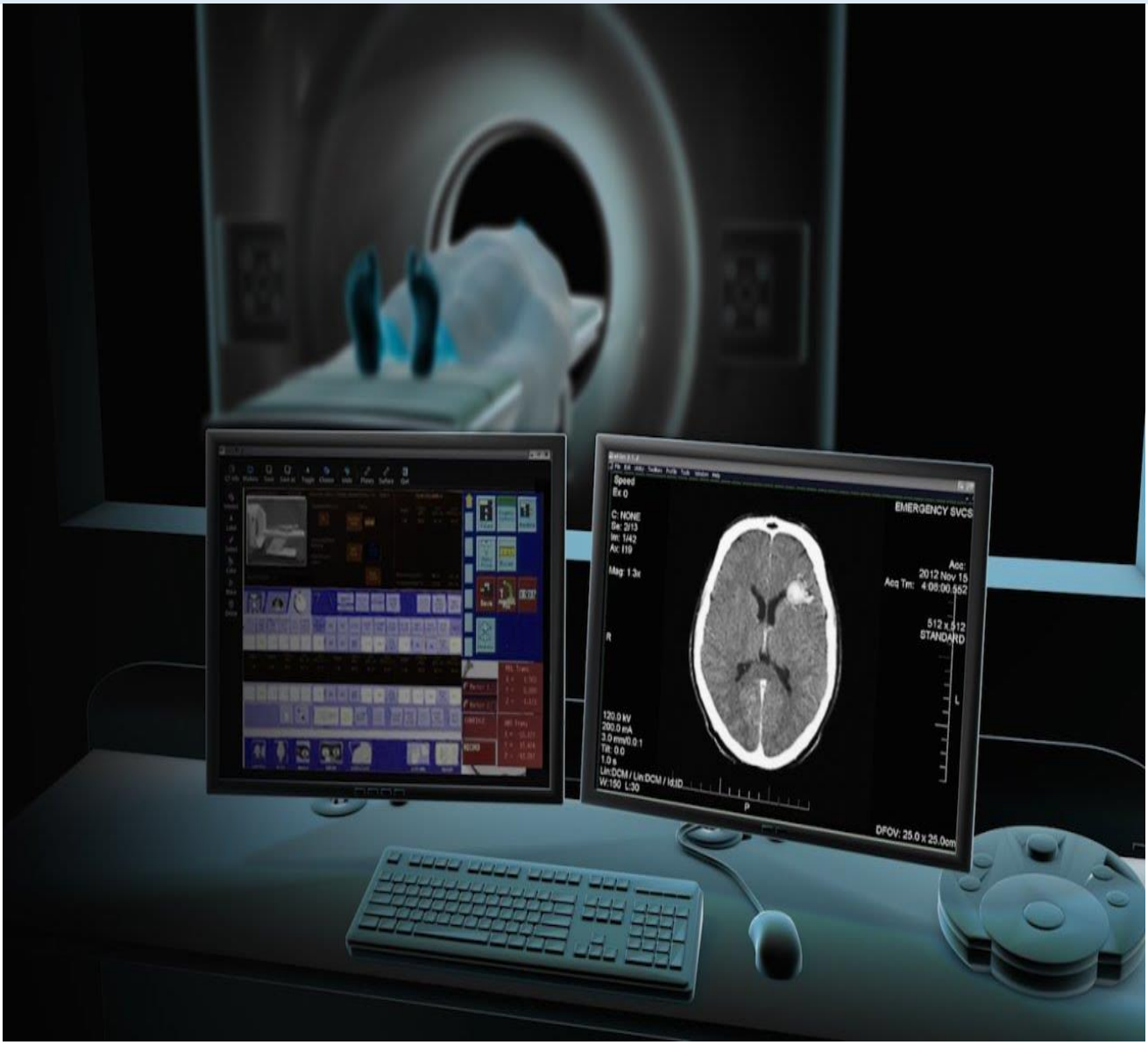
| Simple/Uncomplicated Parapneumonic Effusion | Complicated Parapneumonic Effusion and Empyema |
|---|---|
| ❖ pH >7.2<br>❖ LDH <1/2 serum<br>❖ Glucose >60mg/dL | ❖ ANY one of the following<br>o Large (encompassing more than one-half of the hemi-thorax), free flowing<br>o Effusion of any size with loculations<br>o Thickened parietal pleura on chest CT<br>o Positive-gram stain or culture<br>o pH < 7.20 or glucose < 60 mg/dL |
| ❖ Treat with antibiotics<br>❖ Drainage if necessary, usually they do not required drainage | ❖ **Tube thoracostomy is indicated when pleural fluid glucose is less than 60 mg/dL (less than 3.3 mmol/L) or the pH is less than 7.2**<br>❖ Antibiotic therapy should be continued for at least 2-4 weeks |

**Clinical Pearls:**

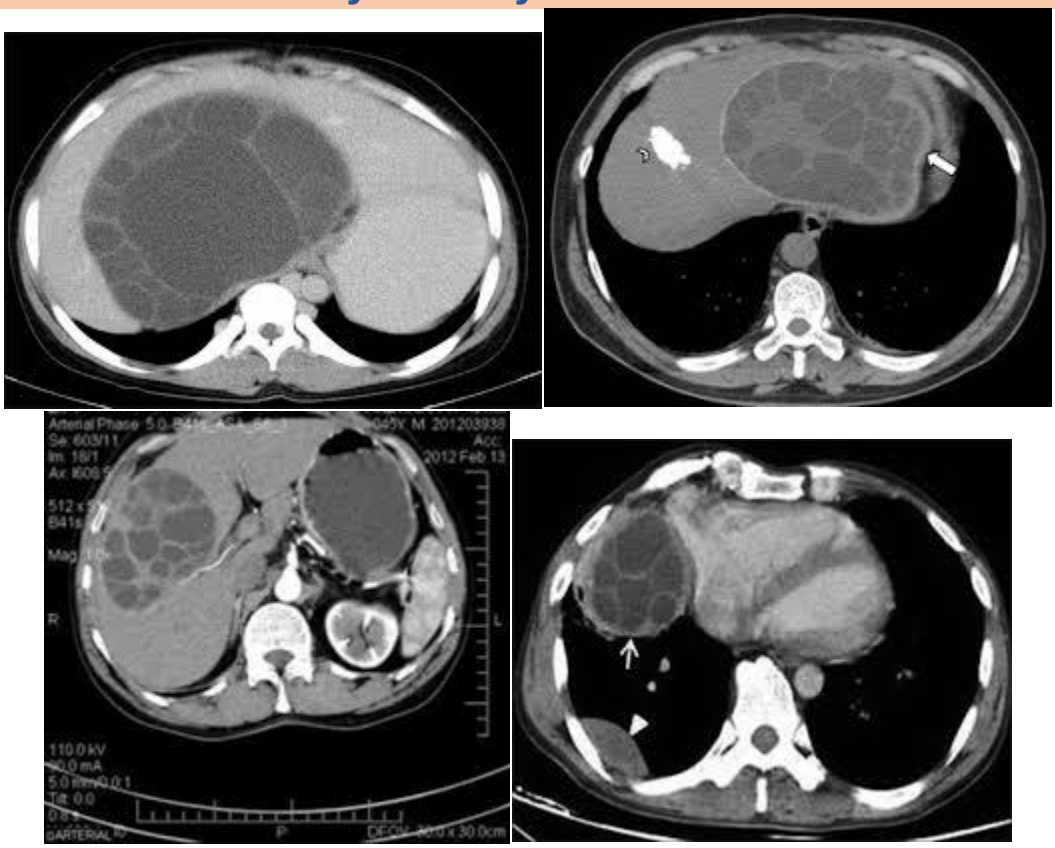
- Empyema--Gross pus in the pleural space or positive gram stain. Positive culture is NOT required for diagnosis
- TB---- Protein > 4.0 g/dL and may exceed 5.0 g/dL

# Station

# CT- Scans



# Hydatid Cyst in Liver



| | |
|---|---|
| **Key findings** | ❖ CT scan of abdomen showing Multiple hypodense areas with multiple septations involving liver |
| **Diagnosis** | ❖ **Hydatid cysts in liver** |

## Toacs Related Question on Hydatid liver disease, Pyogenic liver abscess, Amoebic liver abscess

| | Hydatid liver disease | Pyogenic liver abscess | Amoebic liver abscess |
|---|---|---|---|
| **Causative organism** | ❖ Echinococcus granulosus | ❖ E. coli (most common)<br>❖ Streptococcus milleri<br>❖ Strep. Fecalis & Bacteroides | ❖ Entamoeba histolytica |
| **Pathophysiology** | ❖ it can affect any organ, but liver is the most common followed by lungs, then spleen | ❖ Ascending infection due to biliary obstruction (ascending cholangitis), or spread from empyema of gall bladder | ❖ Trophozites enter portal circulation |
| **Features** | ❖ Cysts in liver (75%), lung , bone and brain | ❖ Multiple abscess | ❖ Single abscess most common in right lobe |
| **Diagnosis** | ❖ Serology<br>✓ Casoni test= 80% positive<br>✓ Indirect hemagglutination test most accurate test<br>❖ CT scan = investigation of choice | ❖ US abdomen<br>❖ Alkaline phosphatase raised | ❖ US abdomen<br>❖ Detectable antibodies in 95% of patient |
| **Treatment** | ❖ **Albendazole 400mg 3 times daily for 30 days**<br>❖ Surgical treatment= ERCP | ❖ **Ampicillin + Ciprofloxacin + Metronidazole**<br>❖ **If penicillin allergic= Ciprofloxacin + clindamycin** | ❖ **Metronidazole + Diloxanide furoate** |

# Station Instruments

# Central Venous Catheter

| | |
|---|---|
| **Introduction & consent** | ❖ Take informed consent<br>❖ Explain procedure and complications |
| **Procedure** | ❖ Position patient head down and towards left side<br>❖ Wash hands<br>❖ Wear gloves<br>❖ Sterilize the area<br>❖ Give inj. Lignocaine cutaneously and subcutaneously around IJV<br>❖ Flush all lumens and lines and clamp all lines<br>❖ Insert syringe into IJV<br>❖ Insert the wire<br>❖ Remove the needle<br>❖ Use scalpel to make small incision into the skin<br>❖ Pass the dilator and wire<br>❖ Remove the dilator and pass CVP line<br>❖ Aspirate and flush all lumens<br>❖ Suture and dressing |
| **Indications** | ❖ IV access<br>❖ Infusion of irritant substances<br>❖ CVP monitoring<br>❖ Inadequate peripheral access<br>❖ Transvenous pacing |
| **Contraindications** | ❖ Obstructed vein (e.g. clot)<br>❖ Stenosis of the vein<br>❖ Severe coagulopathy<br>❖ Contaminated site or burned site<br>❖ Uncooperative awake patient |

**Complications**

| Immediate | Early | Late |
|---|---|---|
| Failure of procedure<br>Pneumothorax<br>Arterial puncture<br>Local hematoma<br>Guidewire-induced arrhythmia | Catheter blockage<br>Chylothorax | Infection<br>Vascular erosion<br>Vessel stenosis<br>Thrombosis |

# Station

# Emergencies, Poisoning & Animal Bites

# Addisonian Crisis

- **A 40-year-old lady presented in OPD with history of generalized weakness, low grade fever and easy fatigability of 4 years. She also reports of decrease in appetite, significant loss of weight with constant nausea over the past 16 months. She also insists that her skin color was fair which have recently changed to black over the body sparing hands and feet. Her past family history is insignificant**.
  - Vital signs temperature 99 F, pulse 88/min. BP 85/50 mmHg, respiratory rate 22/min.
  - Dark brown pigmentation is present all over the body, especially over the elbows and skin creases. Rest of the clinical examination is normal.

- **What is the most likely diagnosis?** Adrenal insufficiency.
- **How would you investigate?**
- **Explain how would you manage such a case?**

  ✓ Hint: Low BP + Dark pigmentation of skin (esp. palmar crease)—suggests Primary adrenal insufficiency (Addison's)

- **A 22-year-old male was admitted with complaints of weakness and weight loss for several weeks. His blood pressure is 80/50 mmHg. Investigation reveals**
  - Na: 122, K: 6.3, HCO3: 30mmol/L

- **What is your likely diagnosis?** Addison's disease
- **Mention one important clinical sign:** Pigmentation of skin and mucous membrane

  ✓ Hint: Low sodium, high potassium and low BP plus skin pigmentation is highly suggestive of Addison's disease

| | |
|---|---|
| **Addison's Disease** | ❖ Causes:<br>○ Idiopathic (most common in developed countries)<br>○ Infections e.g. tuberculosis (most common cause overall)<br>○ Metastatic tumor<br>○ Various infections (e.g. HIV)<br>❖ Characteristics<br>○ **Postural Hypotension**<br>○ **Increased pigmentation of skin (especially palmar crease)**<br>○ Serum Na, Cl-, glucose, and $HCO_3$→ decreased<br>○ **Serum potassium→ increased** |
| **Addisonian Crisis** | ❖ Causes<br>○ Infection, trauma, surgery, missed medication.<br>❖ Characteristics<br>○ Shock (tachycardia, peripheral vasoconstriction, severe postural hypotension occasionally with syncope, oliguria, profound muscle weakness, confusion, altered consciousness leading to coma)<br>○ **Hyperkalemia, hyponatremia, hypoglycemia.** |
| **Management of Addisonian Crisis** | ❖ If suspected, treat before biochemical results.<br>❖ Bloods for cortisol and ACTH.<br>❖ U&ES—can have high K+ (check ECG and give calcium gluconate if needed) and low Na+ (salt depletion, should resolve with rehydration and steroids).<br>❖ Hydrocortisone 100mg IV stat then 8 hourly<br>❖ IV fluid bolus, crystalloid or colloid to support BP.<br>❖ Monitor blood glucose: the danger is hypoglycemia.<br>❖ Blood, urine, sputum for culture, then antibiotics if concern about infection<br>❖ Change to oral steroids after 72h if patient's condition good. |

# Organophosphate Poisoning

- **A 30-year-old farmer is brough to emergency department with nausea, vomiting, diarrhea, pin point pupils, fasciculation and unresponsive to deep pain. There is increased salivation and lacrimation**
- What is your likely diagnosis?

| | |
|---|---|
| **Introduction** | ❖ Organophosphates are widely used as insecticides.<br>❖ inhibit cholinesterases, causing accumulation of acetylcholine at nerve endings and neuromuscular junctions |
| **Clinical Features** | Mnemonic: **DUMBELS**<br>❖ **D**iaphoresis & Diarrhea<br>❖ **U**rination<br>❖ **M**iosis (constricted pupil)<br>❖ **B**ronchospasm<br>❖ **E**mesis<br>❖ **L**acrimation &<br>❖ **S**alivation |
| **Management** | ❖ Clear the airway and maintain ventilation if necessary, maintain IV line<br>❖ Wear gloves; remove soiled clothes. Wash skin.<br>❖ Take blood (FBC and serum cholinesterase activity).<br>❖ Give atropine IV 2mg every 10min till full atropinization (skin dry, pulse >70 pupils dilated).<br>❖ Up to 3 days' treatment may be needed.<br>❖ Also give pralidoxime 30mg/kg IVI over 20min, then 8mg/kg/h, max 12g in 24h. |

# Salicylate Poisoning

- **A 21-year-old unmarried girl is brought to the emergency department in coma. She is sweating, afebrile and tachypneic with fine crackles at the lung bases. She has generalized tonic clonic fit, which responded promptly to diazepam. Her Hb is 13.9 gm/dL, TLC 11×109/L, serum K+=3 mmol/L and serum bicarbonate 10mmol/L**

- **What is the most likely diagnosis?** Salicylate poisoning
- **What are three other conditions you would consider in your differential diagnosis?**
  - DKA---acidosis in young patient should raise the suspicion
  - Poisoning with tricyclic antidepressants and amphetamines also shows metabolic acidosis
  - Uremic acidosis--; i.e. due to renal failure she has developed metabolic acidosis and encephalopathy.
- **What four investigations would you carry out?** —Given below
- **Name two treatment modalities for this patient?** Medical treatment and psychiatry assessment

✓ Hint: Young girl tachycardiac with fits/coma and metabolic acidosis—suggests salicylate poisoning

| | |
|---|---|
| **Introduction** | ❖ Uncoupling of oxidative phosphorylation leads to anaerobic metabolism and the production of lactate and heat.<br>❖ Effects are dose-related and potentially fatal:<br>○ 150mg/kg-------mild toxicity<br>○ 250mg/kg-------moderate<br>○ 500mg/kg-------severe toxicity.<br>○ >700mg/L-------potentially fatal. |
| **Clinical Features** | Mnemonic: **ASPIRIN**<br>❖ **A**ltered mental status (Lethargy—coma)<br>❖ **S**weating/ diaphoresis<br>❖ **P**ulmonary edema<br>❖ **I**ncreased Vital signs (Inc Respiration, Inc Temp, Inc Heart rate)<br>❖ **R**inging in ears<br>❖ **I**rritable<br>❖ **N**ausea & Vomiting<br>❖ Patients present initially with respiratory alkalosis due to a direct stimulation of the central respiratory centers and then develop a metabolic acidosis |
| **Management** | ❖ **General Measures**<br>○ Correct dehydration. |

- Keep patient on ECG monitor.
- Consider gastric lavage if a patient has ingested > 500mg/kg body weight in the previous 1hr
- Give activated charcoal 50g to all presenting ≤1h—consider even if delayed presentation----Consider repeat doses (2 further doses of 50g, 4h apart).

❖ **investigations:**
- Paracetamol and salicylate level
- Glucose, U&E, LFT, INR, ABG, HCO3, CBC.
- Salicylate level may need to be repeated after 2h, due to continuing absorption
- Monitor blood glucose 1–2hrly, beware hypoglycemia

❖ **Correct acidosis:**
- If plasma salicylate level >500mg/L (3.6mmol/L) or severe metabolic acidosis, consider alkalinization of the urine, e.g. with 1.5L 1.26% sodium bicarbonate IV over 3h.
- Aim for urine pH 7.5–8.

❖ **Note:**
- Monitor serum K+ as hypokalemia may occur, and should be treated (caution if acute kidney injury—AKI).

❖ **Dialysis**
- May well be needed if salicylate level >700mg/L, and if AKI or heart failure, pulmonary or cerebral edema, confusion or seizures

# Methanol Poisoning

**A 20-year-old laborer was brought in examination department with few hours' history of nausea, vomiting, and abdominal pain and decreased vision after consumption of a drink. On examination he was drowsy and breathing heavily. Pupils were dilated and sluggishly reacting to light. Fundoscopy revealed hyperemia of optic discs and evidence of retinal edema. Rest of the examination was normal. Subsequently his conscious level deteriorated and he developed generalized tonic-clonic fits. Labs ABG's, pH 7.29, PaO2 = 89 mmHg PaCO2 = 30mmHg, HCO3 8 mEq/L**

**What is your diagnosis?**
**How would you manage this case?**

✓ **Hint: Drowsiness, decreased vision, poorly reactive dilated pupils, metabolic acidosis with normal sugar and urea suggests the diagnosis**

| | |
|---|---|
| **Introduction** | ❖ Methanol is used as a solvent and in antifreeze<br>❖ Methylated spirits are a mixture of ethanol and water with about 5 % methanol |
| **Clinical Features** | ❖ Drowsiness, vomiting, abdominal pain, coma<br>❖ **Blurring of vision.**<br>❖ Severe metabolic acidosis<br>❖ Hyperglycemia and ↓serum amylase.<br>❖ Survivors **may be blind from optic nerve damage** and develop Parkinsonian problems |
| **Management** | ❖ Consider gastric lavage if <1hr since ingestion.<br>❖ **Do not give charcoal.**<br>❖ Measure ABG, U&E, HCO 3, Glucose, FBC, LFTs<br>❖ Plasma methanol if possible.<br>❖ Early use of fomepizole or ethanol<br>❖ Use sodium bicarbonate to correct metabolic acidosis (aim for pH 7.5).<br>❖ Give folinic acid (1mg/kg, max 50mg, IV every 6hr for 48hr)<br>❖ In severe poisoning, refer to ICU for hemodialysis and possibly IPPV. |

# Station

# Clinical Scenarios

# Tuberculosis

- **A 20 years old lady presents with history of anorexia, weight loss, grade fever and cough for the last 6 months. She is also having diarrhea and abdominal pain alternating with constipation for the last 4 months. Examination revealed tenderness in right iliac fossa and hepatosplenomegaly. Examination of CVS is unremarkable.**

- **What is the most clinical diagnosis? —**Disseminated TB (Miliary TB)
- **Give four investigation with possible findings and justifications.**
- **How will you manage this case?**

- **A 30 years old man was brought to outpatient by his friend 10 days prior admission. He developed high grade fever with chills and evening rise. He had cough with scanty sputum in last 2 months. Fever did not settle despite various AntiBiotics and antimalarial therapy. He had to miss his annual business trip to south Africa due to his illness. He has never smoked. On examination he was pale, ill looking with temperature 39.2 C, pulse was 120/min, regular, BP was 120/80 mmHg. He had white coated tongue and cervical adenopathy. Chest examination was normal. cardiac auscultation revealed pericardial rub. Other systems were normal.**
- **Investigations:**
  - Hb 108g/dl
  - ESR 60mm in first hour.
  - TLC 5.2 × 109/L
  - Neutrophils 88%
  - Lymphocytes 10%
  - Eosinophil's 2%
- **CXR showed enlarged cardiac shadow with bilateral hilar lymphadenopathy.**

- **List your differential diagnosis in order of priority.**
  - Lymphoma
  - **Tuberculosis**
  - **Sarcoidosis**
  - Infectious mononucleosis
  - HIV seroconversion
  - Toxoplasmosis
  - Syphilis
  - Brucellosis

- **How will you investigate this patient?**
  - following investigations will be sent
    - Sputum for AFB, to look for mycobacteria tuberculosis,
    - Bronchoscopy followed by bronchial washings these washings will be subjected for ZN staining, C/S and malignant cells.
    - In sarcoidosis, there is reversal of CD4 to CD8 ratio.
    - Excision biopsy of cervical lymph nodes. It will show the characteristic histological changes of lymphoma, tuberculosis, sarcoidosis,
    - CT scan chest followed by Ct guided fine-needle aspiration of hilar lymph nodes. This will not only help us to determine the stage of lymphoma but will also enable us to rule out other causes.
    - Ultrasound abdomen to look for hepatosplenomegaly of intra-abdominal lymph nodes.
    - Peripheral smear examination to look for atypical lymphocytes seen in infectious mononucleosis, HIV or toxoplasma
    - VDRL will be done to rule out syphilis.
    - HIV enzyme linked immunosorbent assay.

| | |
|---|---|
| **Introduction** | ❖ Tuberculosis (TB) is caused by infection with Mycobacterium tuberculosis (MTB)<br>❖ Mycobacteria are **acid-fast bacilli (AFB)**<br>❖ Mode of transmission= Respiratory droplets |
| **Pathogenesis** | ❖ Bacilli are inhaled and lodged in the alveoli<br>❖ Bacilli then initiate recruitment of macrophages and lymphocytes<br>❖ Surviving organisms multiply and disseminate via lymphatic's and the blood stream |

<table>
<tr><td></td><td>❖ Macrophages undergo transmission into Epitheloid and Langerhans cells which aggregate with lymphocytes to form granuloma (Caseating granuloma –Hallmark of disease)<br>o Ghon Focus:<br>• It is the primary lesion characterized by aggregation of numerous granuloma in the periphery of lung<br>o Ghon complex:<br>• It refers to combination of calcified primary lesion (i.e. Ghon focus) with lymph node involvement<br>o Ranke's complex:<br>• It is formed when Ghon complex undergoes fibrosis and calcification</td></tr>
<tr><td>Latent Vs. Reactivation Tuberculosis</td><td><u>Latent Tuberculosis</u><br>❖ Means a patient is infected with Mycobacterium tuberculosis, but the patient does not have active tuberculosis.<br><u>Reactivation Tuberculosis</u><br>❖ The majority of TB cases are due to reactivation of latent infection.<br>❖ Factors implicated in the reactivation of latent TB<br>▪ HIV co-infection<br>▪ Immunosuppressant therapy (chemotherapy/monoclonal antibody treatment) including corticosteroids<br>▪ Diabetes mellitus<br>▪ End-stage chronic kidney disease<br>▪ Malnutrition<br>▪ Ageing</td></tr>
<tr><td>Clinical Features</td><td>❖ Constitutional symptoms (fatigue, anorexia, night sweats, weight loss)<br>❖ <u>Pulmonary TB</u><br>▪ Chronic productive cough ± hemoptysis<br>▪ CXR consolidation or cavitation, lymphadenopathy<br>▪ Non-resolving pneumonia despite standard antimicrobial therapy<br>❖ <u>Miliary TB</u><br>▪ Widely disseminated spread especially to lungs, abdominal organs, marrow, CNS<br>▪ CXR: multiple small 2-4 mm millet seed-like lesions throughout lung<br>❖ <u>Extrapulmonary TB</u>
<table>
<tr><td>Lymph nodes</td><td>❖ Most common extra-pulmonary site<br>❖ Most common site= cervical and mediastinal nodes<br>❖ Painless lymphadenopathy<br>❖ Initially mobile, later becomes matted and suppurative</td></tr>
<tr><td>Gastrointestinal disease</td><td>❖ Most common site = Ileocecal region<br>❖ Right iliac fossa Mass<br>❖ Tuberculous peritonitis= abdominal pain, distention, ascitic fluid exudative with predominance of lymphocytes</td></tr>
<tr><td>Pericardial disease</td><td>❖ Pericardial effusion= raised JVP, increased pericardial dullness<br>❖ Constrictive pericarditis = raised JVP, Early third heart sound</td></tr>
<tr><td>CNS</td><td>❖ Meningitis<br>❖ Tuberculoma</td></tr>
<tr><td>Bone and joint disease</td><td>❖ Bone:<br>o Most common site is spine known as Pott's disease<br>o Most common site of spine= Lower thoracic and lumber region<br>o Psoas abscess = Cold abscess in inguinal region<br>❖ Joints: Most common site= Hip and Knee, Poncet's disease</td></tr>
</table></td></tr>
</table>

❖ **<u>Investigations:</u>**

o **<u>Screening for latent TB</u>**

▪ <u>PPD/Mantoux skin tests/tuberculin skin test.</u>

- 0.1 mL of purified protein derivative (PPD) is injected intradermally on the volar surface (portion of the forearm that is on the same side as the palm)
- The transverse width in millimeters of induration (not erythema) at the skin test site is measured after 48-72 hrs.
- PPD test only useful in latent Tb (asymptomatic patients) and not to be used in symptomatic patients or those with abnormal CXR
- Interpretation:

| | |
|---|---|
| Induration of >15mm | ❖ Considered positive in persons with no risk factors for TB |
| Induration of >10mm | ❖ Considered positive in<br>❖ Prisoners<br>❖ Health care workers<br>❖ Persons working in Nursing homes, Homeless shelters & other health care facilities<br>❖ Persons with following medical conditions (DM, Leukemia, Lymphoma, CKD, CA head & Neck)<br>❖ IV drug users |
| Induration of >5mm | ❖ Considered positive in<br>❖ Recent contacts of individuals with active tuberculosis.<br>❖ HIV positive patients<br>❖ Immunocompromised patient (e.g. Prednisolone 15mg/day for 1 month or more)<br>❖ Persons with fibrotic changes on chest films suggestive of prior tuberculosis.<br>❖ Patients with organ transplants |
| False negative result | ❖ Severe TB<br>❖ New-born and elderly<br>❖ HIV if CD4 count < 200<br>❖ Malnutrition<br>❖ Malignancy |

- **TB blood test "aka" IFN-γ release assay (IGRA)—**Includes (including the QuantiFERON and T-SPOT tests)
  - In patients previously infected with TB, T-cells produce increased amounts of IFN-γ when re-exposed to TB antigen
  - Detects antigen not present in the BCG vaccine or in most types of non-tuberculous mycobacteria (NTM), therefore fewer false positives and superior to older Mantoux tests

- **How to deal with Latent TB**
  - If the latent Tb is proven
  - First exclude active TB (CXR or sputum collection or both)
  - If no active disease—Latent TB proven—and has risk factors for reactivation as described earlier – start on INH 300mg orally –for 9 months

- **Diagnostic test/ investigations for active pulmonary TB**
  - CXR:
  - Sputum staining & Culture:
    - Three morning specimens recommended of sputum (either spontaneous or induced) should be collected for acid-fast bacilli smear and culture.

  - Sputum Gene-Xpert (Nucleic acid amplification (NAA)
    - Detects the DNA in TB bacteria.
    - It uses a sputum sample and can give a result in less than 2 hours.
    - It can also detect the genetic mutations associated with resistance to the drug Rifampicin More

  - Standard drug susceptibility testing of sputum culture

- Considered when a treatment regimen is failing, and when sputum cultures remain positive after 2 months of therapy.

❖ **Treatment:**
- Initial therapy ---(2 months---4 drugs)
  - Mnemonic RIPE: (Rifampicin, Isoniazid, Pyrazinamide, Ethambutol)
- Continuation phase – (4 months—2 drugs)
  - That are Isoniazid & Rifampicin

- Extended treatment: (For 9-12 months)—considered in the following
  - HIV- positive patients, Tuberculous osteomyelitis, Miliary TB, Meningitis (Minimum 12 months), Pregnancy

- INH caused neuropathy
  - Pyridoxine (vitamin B6) 25 to 50 mg PO daily should be used as prophylaxis with INH to prevent neuropathy
  - While 50–100 mg orally daily as treatment if neuropathy develops.

- Baby can be breastfed while taking ATT

- **Drug dosages & Complications**

| Drug | Dosage | DOTS (Directly observe therapy short-course) Initially daily therapy for 2 weeks followed by drugs 3 times/week | Complications |
|---|---|---|---|
| Isoniazid | 5 mg/kg | 15 mg/kg | Peripheral neuropathy, Hepatitis |
| Rifampicin | 10 mg/kg | 10mg/kg | Orange-red color body secretions (urine, tears), hepatitis, |
| Ethambutol | 15-25mg/kg | 25-30mg/kg | Optic neuritis (color blindness for green, decreased visual acuity) |
| Pyrazinamide | 15-30 mg/kg | 50-70 mg/kg | Hyperuricemia, Gout, Hepatitis |
| Streptomycin | 15mg/kg | 25-30mg/kg | 8$^{th}$ (vestibular) nerve damage, nephrotoxicity. |

## Categories of Anti-tuberculosis Drugs: WHO

| | |
|---|---|
| Group 1 (First line drugs) | Isoniazid, Rifampicin, Ethambutol, Pyrazinamide |
| Group 2 (Injectable agents) | Kanamycin, Amikacin, Capreomycin, Streptomycin |
| Group 3 (Fluoroquinolone) | Levofloxacin, Moxifloxacin, Ofloxacin |
| Group 4 (Oral bacteriostatic agents) | Ethionamide, Cycloserine, Para-Aminosalicyclic acid (PAS), Prothionamide |
| Group 5 (Unclear role) | Clofazamine, Linezolid, Amoxicillin/Clavulanate, Imipenem/cilastatin, High dose isoniazid, Clarithromycin, Bedaquiline |

## Multi-Drug Resistant TB (MDR-TB)

❖ TB which is resistant to isoniazid and rifampicin
❖ How to treat MDR-TB (Regimen)

| | | |
|---|---|---|
| Step 1 | Choose any of the following Injectable (Group 2 drugs) | Kanamycin<br>Amikacin |
| Step 2 | Choose a higher generation fluoroquinolone (Group 3 drugs) | Levofloxacin<br>Moxifloxacin |
| Step 3 | Add group 4 Drugs | Cycloserine/Terizidone<br>Para-Aminosalicyclic acid (PAS)<br>Ethionamide/Prothionamide |

- Note:
  - Add two or more Group 4 drugs
  - Ethionamide/Prothionamide considered the most effective
  - Always use a higher generation fluoroquinolone (ciprofloxacin is not effective and never use it)
  - Intensive phase for 8 months (includes injectable) --- and a total of 20 months therapy is advised

## Extensive Drug Resistant TB (XDR-TB)

- TB which is resistant to isoniazid + rifampicin (MDR-TB) + any of the fluoroquinolone (such as levofloxacin or Moxifloxacin) and to at least one of the three injectable second-line drugs (Amikacin, capreomycin or kanamycin).
- How to treat XDR-TB

| | |
|---|---|
| Step 1 | Use pyrazinamide and any other Group 1 agent that may be effective. |
| Step 2 | Use an injectable agent to which the strain is susceptible and consider an extended duration of use (12 months or possibly the whole treatment). If resistant used an agent that has used never before |
| Step 3 | Use a higher-generation fluoroquinolone such as Moxifloxacin or Gatifloxacin |
| Step 4 | Use all Group 4 agents that have not been used extensively in a previous regimen or any that are likely to be effective. |
| Step 5 | Add two or more Group 5 drugs (consider adding bedaquiline) |

Note:
Intensive phase for 8 months (includes injectable) --- and a total of 20 months therapy is advised

## How to Follow Up a Patient of TB

- Conversion of cultures from positive to negative is the most reliable indicator of response to treatment.
- For smear-positive pulmonary TB patients treated with first-line drugs, sputum smear microscopy may be performed at completion of the intensive phase of treatment
- if the specimen obtained at the end of the intensive phase (month 2) is smear-positive, continue with the intensive phase, sputum smear microscopy should be obtained at the end of the third month
- if the specimen obtained at the end of month 3 is smear-positive, sputum culture and drug susceptibility testing (DST) should be performed

# Nephrotic Syndrome

- **A 7-year-old girl presented with swelling of the whole body with scanty micturition for 10 days. Her urine examination reveals color: straw, albumin +++, RBC: nil, pus cells 0-2/HPF, fatty cast present.**

- **What is likely diagnosis?** Nephrotic syndrome
- **Mention investigations?** Serum total protein, albumin, 24 hours urinary protein, serum lipid profile
- **Discuss treatment option?** Prednisolone

✓ Hint: Generalized edema with massive proteinuria is suggestive of nephrotic syndrome

## Nephrotic Syndrome

- Includes a group of conditions characterized by increased basement membrane permeability
- Characteristic features:
  - Massive proteinuria (daily loss of ≥ 3.5 grams of protein per day).
  - Hypoalbuminemia (serum albumin less than 3 g/100 mL)
  - Generalized edema.
  - Hyperlipidemia and hypercholesterolemia are caused by increased hepatic lipoprotein synthesis.
- Causes:
  - **Primary glomerular disease**: Minimal change disease, Membranous GN, Focal segmental glomerulosclerosis
  - **Systemic diseases**: SLE, Amyloidosis, Diabetic nephropathy

| | |
|---|---|
| **Minimal change disease (lipoid Nephrosis)** | ❖ **Most common cause of Nephrotic syndrome in children**<br>❖ Light microscopy → normal-appearing glomeruli.<br>❖ Electron microscopy → disappearance or fusing of epithelial foot processes.<br>❖ **Excellent response to corticosteroids**<br>❖ Associations:<br>○ Hodgkin's lymphoma, thymoma, infectious mononucleosis |
| **Membranous nephropathy (membranous glomerulonephritis)** | ❖ **Most common cause of Nephrotic syndrome in adults**<br>❖ Light microscopy → diffuse capillary and GBM thickening<br>❖ Electron microscopy → spike and dome" appearance with Subepithelial deposits.<br>❖ Poor response to corticosteroids<br>❖ Associations:<br>○ **SLE (10%), hepatitis B**, syphilis, malaria infection; drugs (gold salts or penicillamine); or **malignancy.**<br>○ The disorder sometimes causes renal vein thrombosis, because of loss of antithrombin III, protein C and S & ↑ fibrinogen |
| **Focal segmental glomeroloscelorosis** | ❖ It is more common in African Americans and is **associated with HIV.**<br>❖ It is characterized by sclerosis of some glomeruli, in these affected glomeruli only a portion of capillary tuft is involved<br>❖ Light Microscopy→segmental sclerosis<br>❖ Electron Microscopy→ effacement of foot process/Podocyte effusion (same as Minimal change disease, but minimal change > focal segmental sclerosis) |
| **Diabetic nephropathy** | ❖ ↑ in mesangial matrix → two patterns:<br>○ Diffuse glomerulosclerosis → diffusely increase in mesangial matrix.<br>○ Nodular glomerulosclerosis → nodular accumulations of mesangial matrix material (**Kimmelstiel-Wilson nodules**). |
| **Renal Amyloidosis** | ❖ Amyloidosis refers to accumulation of insoluble fibrillar proteins that form β-pleated sheaths, two types<br>❖ Light Microscopy—**Congo red stain shows apple-green birefringence** |

| Primary (AL) amyloidosis | Secondary (AA) amyloidosis |
|---|---|
| Most common → in developed world. | Less common in developed countries |
| Due to deposition of proteins from Ig **L**ight chains | Occurs in patients with long-standing neoplasia or inflammation and is associated with serum amyloid protein called **AA** protein |
| Can occur as a plasma cell disorder or associated with multiple myeloma and Waldenström macroglobulinemia | It is often seen in concert with tuberculosis, leprosy, RA |

# Nephritic Syndrome (Acute Glomerulonephritis)

- **A 12-year-old boy presents with scanty micturition and puffy face. Urine examination shows color smoky, pus cell 0-2/HPF, RBC 20-30/HPF, RBC cast: present**

- **What is likely diagnosis?** Acute glomerulonephritis
- Mention investigation and treatment? —Given Below
- **Mention two complications?** Acute renal failure, hyperkalemia, hypertensive encephalopathy

  ✓ Hint: scanty micturition and puffy face, mostly after a sore throat is suggestive of acute post streptococcal glomerulonephritis

## Nephritic Syndrome

- (Nephr**I**tic) **I**nflammatory rupture of the glomerular capillaries, with resultant bleeding
- Characterized features: (**A HOPE**)
  - **A**zotemia, **H**ematuria, **H**ypertension, **O**liguria, **P**roteinuria (less than 3g/day), **E**dema

### Acute post streptococcal glomerulonephritis

- Most common type of post-infectious glomerulonephritis in childrens
- Occurs 1–4 weeks after a sore throat caused by group A β-hemolytic streptococci i.e. streptococcus pyogenes
- Type III hypersensitivity reaction.
- Clinical features:
  - Sudden onset of fever, oliguria, *hematuria (cocoa-colored urine)*
- Findings:
  - Serum C3 decreased
  - ASO titers elevated
  - Electron microscopy→ Subepithelial humps
  - Immunofluorescence→ lumpy bumpy appearance
- Management:
  - Symptomatic: fluid and sodium restrictions; loop diuretics for HTN and edema
  - In severe cases, may require dialysis if renal function significantly impaired
  - Treat with penicillin or erythromycin if evidence of persistent GAS infection

### Rapidly progressive (crescentic) glomerulonephritis

- Nephritic syndrome that progresses rapidly to renal failure within weeks or months
- Light microscopy & Immunofluorescence → *Crescent shape glomerulonephritis*
- Disease processes that may result in this pattern are

| Goodpasture syndrome | Wegener's glomerulonephritis |
|---|---|
| ○ Involves lung and renal vessels | ○ Involving upper respiratory tract, lung and renal vessels |
| ○ Hemorrhagic pneumonitis (pneumonia plus hemoptysis)<br>○ Nephritic syndrome | ○ Sinusitis, otitis media<br>○ Hemorrhagic pneumonitis (pneumonia plus hemoptysis)<br>○ Nephritic syndrome |

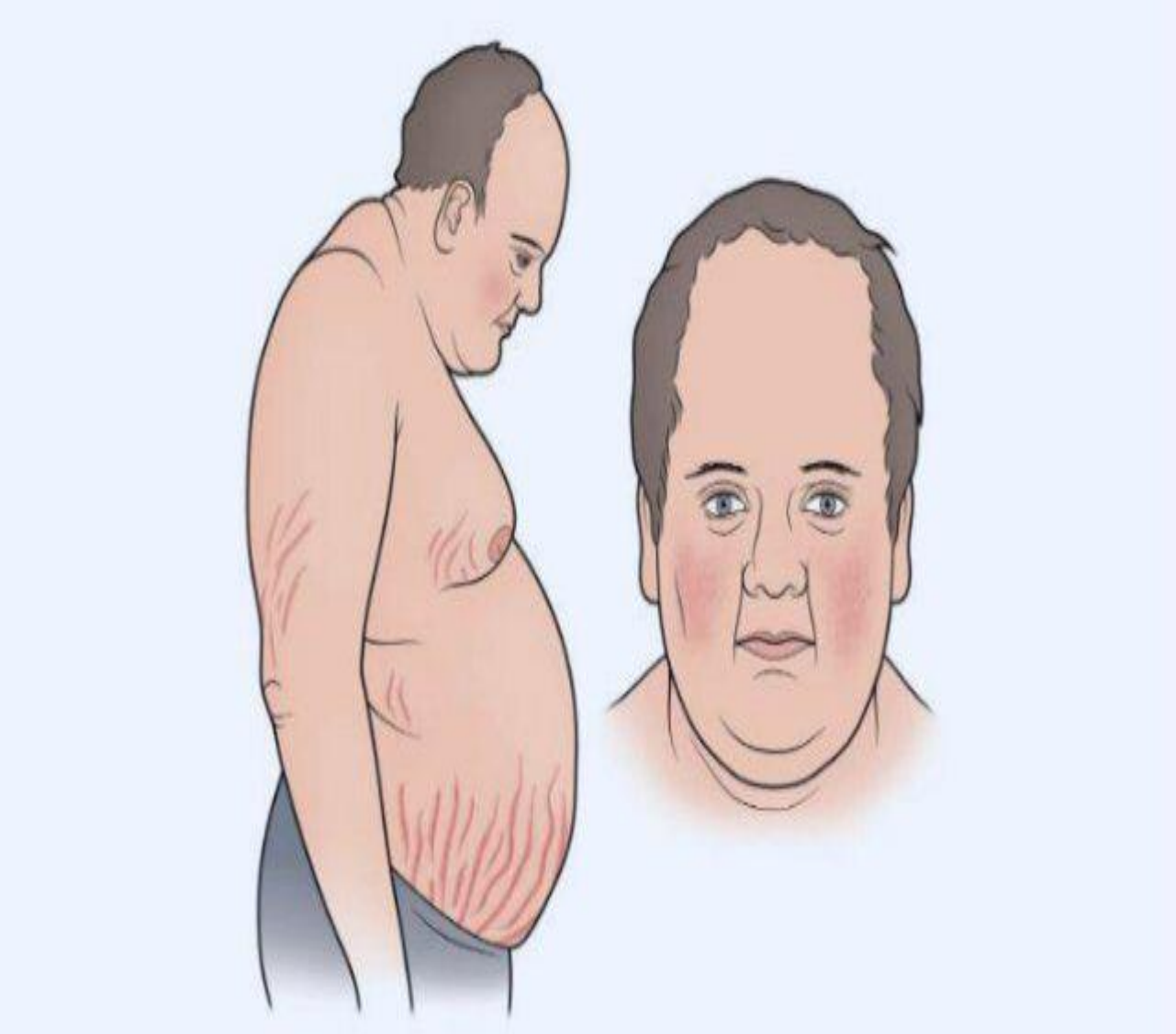
### IgA nephropathy (Berger disease)

- *Most common type of nephritic syndrome overall and is due to deposition of IgA in the mesangium*
- *It presents with recurrent episodes of hematuria following upper RTI, GI infections, occurs 1-2 days after infection*
- Associations: *coeliac disease/dermatitis herpetiformis,* ***Henoch-Schonlein purpura***
- Light microscopy→ mesangial expansion
- Immunofluorescence→ granular mesangial IgA and lambda light chain deposition
- *Not to be confused with Buerger disease (Thromboangiitis obliterans).*

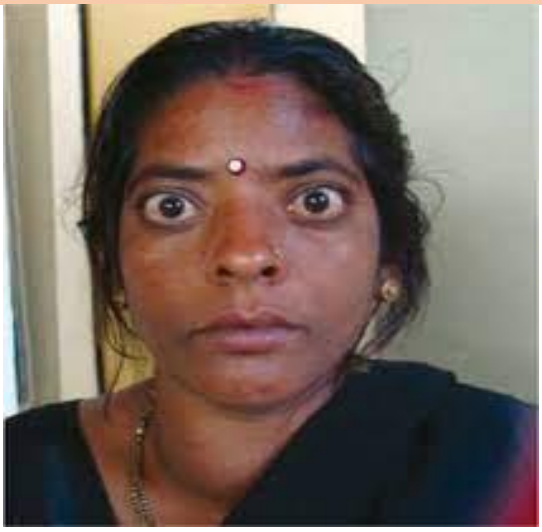
### Alport syndrome

- ***Most commonly X-linked dominant.***
- *Defective glomerular basement membrane synthesis due to abnormal collagen type IV*
- Clinical features: Mnemonic: "**can't see, can't pee, and can't hear a bee**."
  - Eye problems (e.g., retinopathy, lens dislocation)
  - Glomerulonephritis
  - Sensorineural deafness
- Electron microscopy→*"Basket-weave" appearance.*

# Station Pictograms

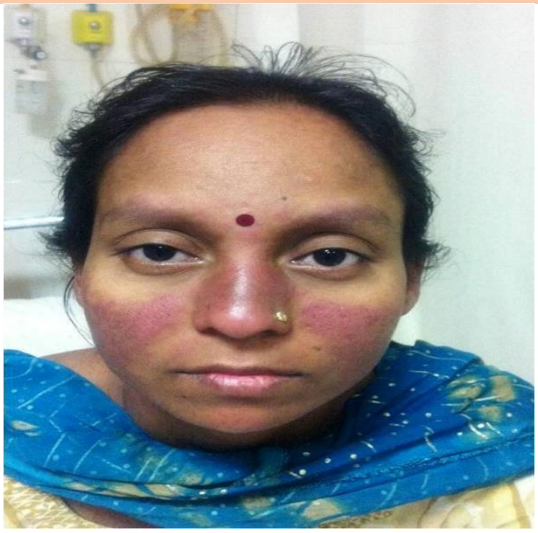


# Graves Disease



| | |
|---|---|
| **Identify the picture** | ❖ Bilateral exophthalmos<br>❖ Diffuse goitre<br>❖ Anxious look<br>❖ My diagnosis is Graves diseases |
| **Features** | ❖ Typical features of thyrotoxicosis<br>❖ Specific signs limited to Grave's:<br>○ Eye signs (30% of patients): exophthalmos, ophthalmoplegia<br>○ Pretibial Myxedema<br>○ Thyroid acropachy: |
| **Autoantibodies** | ❖ Anti-TSH receptor stimulating antibodies (90%)<br>❖ Anti-thyroid peroxidase antibodies (50%) |
| **Management** | ❖ **Treatment options include:**<br>○ **Titration of anti-thyroid drugs (ATDs, for example carbimazole)**<br>✓ Carbimazole is started at 40mg and reduced gradually to maintain euthyroidism<br>✓ Typically continued for 12-18 months<br>✓ Patients following an ATD titration regime have been shown to suffer fewer side effects than those on a block-and-replace regime<br>✓ The major complication of carbimazole therapy is agranulocytosis<br>○ **Block-and-replace regimes:**<br>✓ Carbimazole is started at 40mg<br>✓ Thyroxine is added when the patient is euthyroid<br>✓ Treatment typically lasts for 6-9 months<br>○ **Radioiodine treatment**<br>✓ Goiter shrinkage may occur in up to 30% following RAI.<br>✓ Contraindications include<br>▪ Pregnancy (should be avoided for 4-6 months following treatment) and<br>▪ Age < 16 years.<br>▪ Thyroid eye disease is a relative contraindication, as it may worsen the condition<br>○ **Surgery**<br>✓ The surgical procedure of choice for patients with Graves' disease is a total resection of one lobe and a subtotal resection of the other lobe, leaving about 4 g of thyroid tissue (Hartley–Dunhill operation).<br>○ **Propranolol is often given initially to block adrenergic effects** |
| **Thyroid eye disease** | ❖ **Prevention:**<br>○ Smoking is the most important modifiable risk factor for development of thyroid eye<br>❖ **Disease**<br>○ Topical lubricants may be needed to help prevent corneal inflammation caused by exposure<br>○ Rapid administration of steroids<br>○ Where sight is threatened, orbital decompression may be necessary |

# Systemic Lupus Erythematosus (SLE)



| | |
|---|---|
| **Identify the picture** | ❖ Skin rash---butterfly distribution<br>❖ My diagnosis is SLE |
| **SLE** | ❖ Most common connective tissue disorder, **more common in women's**<br>❖ It is multisystem inflammatory autoimmune disorder<br>❖ Classic scenario is like rash, joint pain, and fever, commonly in a female of reproductive age<br>❖ Two most important lesions frequently asked in exam:<br>○ **L**ibman-**S**acks **E**ndocarditis— (Mnemonic: (**LSE** in **SLE**)<br>▪ Nonbacterial, thrombi usually on mitral or aortic valve<br>○ Lupus nephritis:<br>▪ Glomerular deposition, can be nephritic or nephrotic |
| **SLE and Pregnancy:** | ❖ Unlike many autoimmune diseases systemic lupus erythematous (SLE) often becomes worse during pregnancy and the puerperium<br>❖ Neonatal complications include congenital heart block, it is strongly associated with anti-Ro (SSA) antibodies |
| **Findings:** | **Antinuclear antibodies (ANA)** — **Sensitive**, not specific<br>**Anti-dsDNA antibodies** — **Highly specific**, poor prognosis (renal disease)<br>**(Anti-Smith antibodies** — **Most specific**, not prognostic<br>**Antihistone antibodies** — Sensitive for drug-induced lupus (eg, hydralazine, procainamide)<br>**↓C3, C4** — Formation of complexes leads to consumption of complement |

❖ **Diagnostic criteria (manifestation)**

| **Presence Of ≥ 4 Of Following 11 Criteria (mnemonic: DOPAMINe RASH)** |
|---|
| **D**iscoid rash |
| **O**ral ulcers |
| **P**hotosensitivity |
| **A**rthritis (non-erosive) |
| **M**alar rash (**butterfly rash on cheeks and nose with sparing of nasolabial folds**) |
| **I**mmunological → positive anti-dsDNA (very specific, prognostic), anti-Sm (very specific, non prognostic), antiphospholipid antibodies |
| **N**eurological: seizures or psychosis |
| **R**enal: proteinuria, glomerulonephritis |
| **A**NA positive (Best screening test) |
| **S**erositis: Pericarditis, Pleuritis |
| **H**aematological: hemolytic anemia, lymphopenia, leukopenia, thrombocytopenia |

❖ **Treatment:** NSAIDs, steroids, immunosuppressants, hydroxychloroquine.